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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 12:08:39 ON 09 NOV 2006

=> file reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'REGISTRY' ENTERED AT 12:08:59 ON 09 NOV 2006


=> e p21-activated kinase 4/cn
E1            3     P21-ACTIVATED KINASE 3 (PAN TROGLODYTES VERUS CLONE C3 GENE
                    PAK3)/CN
E2            1     P21-ACTIVATED KINASE 3 (PONGO PYGMAEUS GENE PAK3)/CN
E3            1 --> P21-ACTIVATED KINASE 4/CN
E4            1     P21-ACTIVATED KINASE 5/CN
E5            1     P21-ACTIVATED KINASE 7 (HUMAN CLONE MGC:26232 IMAGE:4821164)
                    /CN
E6            1     P21-ACTIVATED KINASE PAK65/CN
E7            1     P21-ACTIVATED PROTEIN KINASE/CN
E8            1     P21-ACTIVATED PROTEIN KINASE (CAENORHABDITIS ELEGANS CLONE Y
                    K116F6)/CN
E9            1     P21-ACTIVATED PROTEIN KINASE (HUMAN CLONE 21 GENE PAK1)/CN
E10           1     P21-ACTIVATED PROTEIN KINASE (HUMAN CLONE 212 GENE PAK2)/CN
E11           1     P21-ACTIVATED PROTEIN KINASE 1/CN
E12           1     P21-ACTIVATED PROTEIN KINASE 3/CN

=> s e3
L1            1 "P21-ACTIVATED KINASE 4"/CN


=> e map kinase kinase 7/cn
E1            1     MAP KINASE KINASE 6 (CARP)/CN
E2            1     MAP KINASE KINASE 6 (HUMAN GENE MKK6)/CN
E3            1 --> MAP KINASE KINASE 7/CN
E4            1     MAP KINASE KINASE 7 (ARABIDOPSIS THALIANA GENE BUD1/AT1G1835
                    0)/CN
E5            1     MAP KINASE KINASE 7 (HUMAN GENE MKK7)/CN
E6            1     MAP KINASE KINASE 7 (MUS MUSCULUS ISOENZYME B)/CN
E7            1     MAP KINASE KINASE 7 (MUS MUSCULUS STRAIN CD-1)/CN
E8            1     MAP KINASE KINASE ANQ1 (ARABIDOPSIS THALIANA GENE ANQ1/ATMKK
                    6)/CN
E9            1     MAP KINASE KINASE DDMEK1 (DICTYOSTELIUM DISCOIDEUM STRAIN KA
                    X3 GENE MEKA)/CN
E10           1     MAP KINASE KINASE KINASE/CN
E11           1     MAP KINASE KINASE KINASE (CAENORHABDITIS ELEGANS GENE NSY-1)
                    /CN
E12           1     MAP KINASE KINASE KINASE (CRYPTOCOCCUS NEOFORMANS NEOFORMANS
                     STRAIN JEC21)/CN

=> s e3
L2            1 "MAP KINASE KINASE 7"/CN

=> set exp cont
SET COMMAND COMPLETED

=> sel l1 chem
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E13 THROUGH E17 ASSIGNED

=> sel l2 chem
E18 THROUGH E34 ASSIGNED


=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      12.82      13.03

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
       AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
       CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,
       DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 12:12:45 ON 09 NOV 2006

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s e13-17 and e18-34
   3 FILES SEARCHED...
   6 FILES SEARCHED...
         1   FILE BIOSIS
  10 FILES SEARCHED...
  12 FILES SEARCHED...
  13 FILES SEARCHED...
         4   FILE CAPLUS
  15 FILES SEARCHED...
  19 FILES SEARCHED...
  21 FILES SEARCHED...
  23 FILES SEARCHED...
  25 FILES SEARCHED...
  27 FILES SEARCHED...
         1   FILE EMBASE
  29 FILES SEARCHED...
  30 FILES SEARCHED...
  34 FILES SEARCHED...
         2   FILE GENBANK
         1   FILE IFIPAT
  37 FILES SEARCHED...
  41 FILES SEARCHED...
  44 FILES SEARCHED...
  48 FILES SEARCHED...
  53 FILES SEARCHED...
  55 FILES SEARCHED...
  58 FILES SEARCHED...
         3   FILE TOXCENTER
         1   FILE USPATFULL
  61 FILES SEARCHED...
  63 FILES SEARCHED...
  66 FILES SEARCHED...

   7 FILES HAVE ONE OR MORE ANSWERS,   68 FILES SEARCHED IN STNINDEX

L3   QUE ("PAK4 KINASE"/BI OR "PROTEIN KINASE PAK4"/BI OR "P21-ACTIVATED KINASE
          4"/BI OR "P21-ACTIVATED PROTEIN KINASE 4"/BI OR 220064-77-7/BI) AND (
         "GENE C-JUN PROTEIN KINASE N-TERMINAL KINASE 2"/BI OR "JNNK2 KINASE"/B
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          I OR "JUN N-TERMINAL KINASE KINASE 2"/BI OR "KINASE (PHOSPHORYLATING),
           GENE C-JUN PROTEIN KINASE N-TERMINAL KINASE, 2"/BI OR "MAP KINASE KIN
          ASE 7"/BI OR MAP2K7/BI OR "MEK7 KINASE"/BI OR "MEK7 PROTEIN KINASE"/BI
           OR "MITOGEN-ACTIVATED PROTEIN KINASE KINASE 7"/BI OR "MKK7 KINASE"/BI
           OR "MKK7 PROTEIN KINASE"/BI OR MKK7/BI OR "PROTEIN KINASE JNKK2"/BI O
          R "PROTEIN KINASE MEK7"/BI OR "PROTEIN KINASE MKK7"/BI OR 198228-69-2/
          BI OR 335605-46-4/BI)

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COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                20.74      33.77

FILE 'CAPLUS' ENTERED AT 12:33:18 ON 09 NOV 2006
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FILE 'USPATFULL' ENTERED AT 12:33:18 ON 09 NOV 2006
CA INDEXING COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

=> s l3
   1 FILES SEARCHED...
   3 FILES SEARCHED...
   5 FILES SEARCHED...
L4          14 L3

=> dup rem l4
DUPLICATE IS NOT AVAILABLE IN 'GENBANK'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L4
L5             9 DUP REM L4 (5 DUPLICATES REMOVED)
                 ANSWERS '1-4' FROM FILE CAPLUS
                 ANSWERS '5-6' FROM FILE GENBANK
                 ANSWER '7' FROM FILE BIOSIS
                 ANSWER '8' FROM FILE IFIPAT
                 ANSWER '9' FROM FILE USPATFULL

=> d bib abs hit 1-4 7-9

L5   ANSWER 1 OF 9  CAPLUS  COPYRIGHT 2006 ACS on STN  DUPLICATE 1
AN   2006:513522  CAPLUS  Full-text
DN   145:21119
TI   Harnessing network biology to improve drug discovery
IN   Macdonald, Marnie L.; Westwick, John K.; Keon, Brigitte; Lamerdin, Jane;
     Michnick, Stephen W.
```

PA Odyssey Thera, Inc., USA
SO PCT Int. Appl., 115 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006058014	A2	20060601	WO 2005-US42344	20051122
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	US 2006160109	A1	20060720	US 2005-282745	20051121
PRAI	US 2004-629558P	P	20041122		
	US 2005-282745	A	20051121		

AB This invention provides principles, methods and compns. for ascertaining the mechanism of action of pharmacol. important compds. in the context of network biol., across the entire scope of the complex pathways of living cells. Importantly, the principles, methods and compns. provided allow a rapid assessment of the on-pathway and off-pathway effects of lead compds. and drug candidates in living cells, and comparisons of lead compds. with well-characterized drugs and toxicants to identify patterns associated with efficacy and toxicity. The invention will be useful in improving the drug discovery process, in particular by identifying drug leads with desired safety and efficacy and in effecting early attrition of compds. with potential adverse effects in man.

IT 9001-62-1, Lipase 9003-98-9, DNAse 9076-57-7, Histone deacetylase 63551-76-8, Phospholipase C γ 90698-26-3, p70S6 Kinase 115926-52-8 116283-83-1, EEF-2 kinase 137632-07-6, Erk1 kinase 137632-08-7, Erk2 kinase 139691-76-2, c-Raf kinase 140208-22-6, Cdc25A phosphatase 141349-86-2, Cdk2 kinase 141436-78-4, Protein kinase Cα 141467-20-1 142805-58-1, Mek1 kinase 143375-65-9, Cdc2 kinase 144114-16-9, Fak kinase 144697-16-5, B-Raf kinase 144697-17-6, c-Src kinase 146702-84-3, MEKK1 kinase 147014-97-9, Cdk4 kinase 148640-14-6, Akt1 kinase 149371-05-1 150316-07-7, MAP3K8 150316-14-6 151821-62-4, Ubiquitin C 152478-56-3, JAK1 kinase 154907-65-0, Chk1 kinase 165245-96-5, Protein p38α kinase 165245-99-8, Gene plk protein kinase 172306-54-6 176023-60-2, Akt2 kinase 177893-51-5, Protein kinase Pak1 182938-07-4, Protein p160ROCK kinase 185464-61-3, Protein kinase MEKK5 191359-13-4 191808-15-8, Pdk1 kinase 192140-83-3, Protein kinase Pak2 192230-91-4, JNKK1 protein kinase 212906-83-7, Protein kinase RIP2 220064-77-7, Protein kinase Pak4 260402-73-1 260402-76-4, Protein kinase Elk1 289898-51-7, JNK1 kinase 289899-93-0, JNK2 kinase 301166-54-1 335605-46-4, Protein kinase JNKK2 362516-16-3, IκB-α Kinase 362517-43-9, IκB-β Kinase 392658-87-6, Protein kinase RIP4 415965-81-0, Prolyl isomerase Pin1 443900-95-6 460751-71-7, IκB-ε Kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(harnessing network biol. to improve drug discovery)

L5 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
AN 2006:318918 CAPLUS Full-text
DN 144:343640
TI Resorcylic acid lactone kinase inhibitors, and their therapeutic use for the treatment of cancers and other conditions
IN Santi, Daniel V.; Reid, Ralph C.; Hutchinson, Richard C.; Sundermann, Kurt F.; Lau, Janice
PA Kosan Biosciences Incorporated, USA
SO PCT Int. Appl., 110 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006036941	A2	20060406	WO 2005-US34537	20050926
	WO 2006036941	A3	20061026		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	US 2006079494	A1	20060413	US 2005-236244	20050926
PRAI	US 2004-613680P	P	20040927		
	US 2004-629575P	P	20041118		
	US 2005-698520P	P	20050711		

OS MARPAT 144:343640
AB Resorcylic acid lactones having a C5-C6 cis double bond and a ketone at C7 and other compds. capable of Michael adduct formation are potent and stable inhibitors of a subset of protein kinases having a specific cysteine residue in the ATP binding site. Compds. of the invention include e.g. hypothemycin. Compound preparation is included.
IT 335605-46-4, Protein kinase MKK7
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(isoform β; resorcylic acid lactone kinase inhibitors, and therapeutic use)
IT 52-90-4, L-Cysteine, biological studies 51845-53-5, Protein kinase Zipk 79079-06-4, EGF receptor kinase 90698-26-3, Protein kinase rsk1 98037-52-6, Abl kinase 103843-29-4, IGF-I receptor tyrosine kinase 114051-78-4, Lck kinase 134549-83-0, Protein kinase STYK1 136396-12-8, PDGF β-receptor kinase 137632-03-2, Met kinase 137632-06-5, Protein kinase Csk 137632-07-6, Erk1 kinase 137632-08-7, Protein kinase MAPK1 138359-29-2, Kit tyrosine kinase 138674-26-7, Syk kinase 139691-76-2, c-Raf kinase 140208-17-9, Lyn kinase 141349-87-3, Fyn kinase 141349-91-9, Yes kinase 141350-03-0, Flt1 kinase 141436-78-4, Protein kinase Cα 141460-90-4, Fes kinase 142008-29-5, Protein kinase A 142805-58-1, Protein kinase MEK1 144114-11-4, Ros kinase 144376-45-4, Pim-1 kinase 144378-32-5, Cyclin B-CDK1 kinase 144638-77-7, Protein kinase flt4 144697-16-5, b-Raf kinase 144697-17-6, c-Src kinase 144941-32-2, Fgr kinase 145539-86-2, Hck

kinase 146279-88-1, CDK2/cyclin A kinase 146279-89-2, CDK2/cyclin E kinase 146279-92-7, Ret kinase 146279-97-2, EphB2 receptor tyrosine kinase 146702-86-5, Type II TGF- receptor serine/threonine kinase 146838-19-9, Arg kinase 146838-30-4, MAPKAP-K2 kinase 147014-96-8, CDK5 kinase 147230-71-5, Flt3 kinase 148047-29-4, Tie2 kinase 148047-34-1, Zap-70 kinase 148640-14-6, Protein kinase B 149146-03-2, FGFR3 tyrosine kinase 149146-91-8, TrkB kinase 149147-12-6, Btk kinase 149433-91-0, EphA2 receptor tyrosine kinase 150027-15-9, FGFR1 tyrosine kinase 150316-14-6, MEK2 kinase 150977-45-0, Kdr kinase 152743-99-2, Gene erbB4 protein kinase 152787-58-1, Protein kinase TrkA 153190-51-3, Brk kinase 153190-60-4, Protein kinase DDR2 153190-63-7, Protein kinase Axl 153190-64-8, Gene Fer protein kinase 153570-69-5, FGFR4 tyrosine kinase 154907-65-0, CHK1 kinase 154907-66-1, Cyclin D-dependent kinase CDK6 154907-68-3, Gene rse protein kinase 155215-87-5, Jnk kinase 156621-09-9, Protein kinase MSK1 160477-87-2, Protein kinase CDKL1 161384-20-9, Protein kinase Cμ 165245-94-3, NEK2 kinase 165245-96-5, Protein kinase SAPK2a 166433-56-3, Alk receptor tyrosine kinase 167397-96-8, IRAK kinase 170780-46-8, Pyk2 kinase 172308-13-3, Protein kinase MEK3 175780-17-3, Protein kinase MAPKAP-K3 176023-64-6, SAPK3 kinase 178037-70-2, Protein kinase Sgk 178303-46-3, Protein kinase Bmx 179800-23-8, Protein kinase SAPK2b 181186-91-4, Plk3 protein kinase 182238-33-1, Ron receptor tyrosine kinase 182372-18-5, Protein kinase MST2 182938-07-4, Rock-I kinase 182938-08-5, Protein kinase rock-II 185156-08-5, Protein kinase prk2 185464-61-3, Ask1 kinase 186359-58-0, Protein kinase ZAK 188596-65-8, NIK kinase 191359-14-5, MKNK2 kinase 191808-15-8, PDK1 kinase 192140-83-3, PAK2 kinase 192230-91-4, MEK4 kinase 192333-55-4, SAPK4 kinase 194739-73-6, Protein kinase MKK6 206138-20-7, Protein kinase Prak 207137-52-8, NLK protein kinase 210419-07-1, Ro 09-2210 212906-83-7, Protein kinase RIPK2 216974-70-8, EphB4 receptor tyrosine kinase 219917-92-7, L 783277 220064-77-7, PAK4 kinase 222838-93-9, Protein kinase ERK8 244634-79-5, CHK2 kinase 253170-37-5, MSK2 kinase 253863-19-3 267008-45-7, Protein kinase MINK 285571-90-6, NEK6 kinase 291756-39-3, JNK3 kinase 294190-69-5, Protein kinase TOPK 321547-59-5, Protein kinase SPEG 327046-95-7, MEK5 kinase 333425-95-9, Protein kinase D2 344300-27-2, Cyclin E-cdk3 kinase 355120-76-2, Protein kinase CDKL3 362516-16-3, IKKα kinase 362517-43-9, IKKβ kinase 366806-33-9, Protein kinase CK2 372092-80-3, Protein kinase 377752-08-4, Protein kinase rsk2 389133-24-8, Protein kinase rsk3 402934-70-7, NEK7 protein kinase 458560-40-2, Aurora-A kinase 472998-88-2, Protein kinase Cι 476196-08-4, Protein kinase CaMKIV 488850-98-2, Protein kinase Cδ 525566-59-0, TAK1 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resorcylic acid lactone kinase inhibitors, and therapeutic use)

L5 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5
AN 2005:902703 CAPLUS Full-text
DN 143:272498
TI Gene expression profiles in the diagnosis and treatment of Alzheimer's disease
IN Landfield, Philip W.; Porter, Nada M.; Chen, Kuey Chu; Geddes, James; Blalock, Eric
PA University of Kentucky Research Foundation, USA
SO PCT Int. Appl., 114 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005076939	A2	20050825	WO 2005-US3668	20050209
	WO 2005076939	A3	20060706		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2004-542281P P 20040209

AB Genes showing altered patterns of expression in the brain that are associated with the neurol. changes found in Alzheimer's disease and that can be used in the early diagnosis of the disease, including the incipient form of the disease, are identified. The methods and kits of the invention utilize a set of genes and their encoded proteins that are shown to be correlated with incipient Alzheimer's disease.

IT Proteins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (Dendrin, gene for, expression of, in diagnosis of Alzheimer's disease; gene expression profiles in diagnosis and treatment of Alzheimer's disease)

IT Cyclins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (H, gene for, expression of, in diagnosis of Alzheimer's disease; gene expression profiles in diagnosis and treatment of Alzheimer's disease)

IT Gene, animal

RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses) (HBA1, expression of, in diagnosis of Alzheimer's disease; gene expression profiles in diagnosis and treatment of Alzheimer's disease)

IT 90119-11-2, Leukotriene B4 ω-hydroxylase 90597-47-0, Peptidylglycine-amidating monooxygenase 90698-26-3, Ribosomal protein S6 kinase 90880-95-8, Proenkephalin 91608-96-7, Interferon-induced double-stranded RNA-dependent protein kinase 94219-29-1, Fatty-acid elongase 95076-93-0, Peptidyl-prolyl isomerase 96282-35-8, Serine proteinase inhibitor 96779-46-3, Mephenytoin 4-hydroxylase 97089-82-2, 6-Pyruvoyltetrahydropterin synthase 99676-46-7, Prohormone convertase 1 102576-81-8 102577-19-5, Neuromedin B 102686-80-6, Nifedipine oxidase 103220-14-0, Corticostatin 104404-69-5, Hephaestin 104645-76-3, Phosphatidylinositol-4-phosphate 5-kinase 105638-50-4, Protein-L-isoaspartate methyltransferase 106283-10-7, Inositol 1,4,5-trisphosphate 3-kinase 109136-49-4, Deubiquitinating enzyme 110639-28-6, Endooligopeptidase A 111694-13-4, Inositol polyphosphate-1-phosphatase 113189-02-9 115926-52-8, Phosphoinositide-3-kinase 115966-66-0, Histatin 1 117698-12-1, Paraoxonase 120038-28-0, Carboxypeptidase M 122544-66-5, Aspartate β-hydroxylase 123082-54-2 125692-40-2, Endothelin 3 125978-95-2, Nitric oxide synthase 130122-81-5, Dopachrome tautomerase 131144-94-0, Pristanoyl-CoA oxidase 133249-50-0, Short chain 3-hydroxyacyl-Coenzyme A dehydrogenase 133249-52-2, Thymine-DNA glycosylase 133876-97-8, Phospholipase A2 137632-08-7, Mitogen-activated protein kinase 1 138069-86-0, APEX nuclease

138238-81-0, Endothelin converting enzyme 1 141349-86-2, Cyclin-dependent kinase 2 141436-78-4, Protein kinase C 141467-21-2, Calcium calmodulin-dependent protein kinase 142008-29-5, CAMP-dependent protein kinase 143180-75-0 143636-96-8, α-Endosulfine 144114-16-9, Protein tyrosine kinase 2 144697-17-6, c-Src tyrosine kinase 146480-35-5, Matrix metalloproteinase 2 146838-30-4, Mitogen-activated protein kinase-activated protein kinase 2 147014-96-8, Cyclin-dependent kinase 5 149147-12-6 149371-03-9, Topoisomerase III 150316-14-6, Mitogen-activated protein kinase kinase 2 150605-49-5, Palmitoyl-protein thioesterase 1 151769-16-3, Metalloproteinase ADAM 17 152166-55-7, Double-stranded RNA specific adenosine deaminase 152478-57-4, Januskinase2 153190-61-5, Tyrosine kinase 2 153190-63-7, AXL receptor tyrosine kinase 153700-57-3, G Protein-coupled receptor kinase 5 156859-16-4, RYK receptor tyrosine kinase 157482-36-5, Janus kinase 3 158129-99-8, G Protein-coupled receptor kinase 6 163441-58-5, MATK tyrosine kinase 163913-61-9, Apolipoprotein B mRNA editing enzyme 165245-96-5, Mitogen-activated protein kinase 14 167397-96-8, IRAK-1 kinase 169592-56-7, Caspase 3, apoptosis-related cysteine protease 169592-62-5, Cyclin-dependent kinase 10 172306-41-1, PCTAIRE protein kinase 1 172306-54-6, LIM kinase 2 172308-13-3, Mitogen-activated protein kinase kinase 3 172521-74-3, Relaxin 1 176023-64-6, Mitogen-activated protein kinase 12 178037-70-2, Serum and glucocorticoid regulated kinase 180189-96-2, Caspase 9 182372-14-1, Caspase 2 182372-15-2, Caspase 6 182762-08-9, Caspase 4 182938-07-4, Rho-associated, coiled-coil containing protein kinase 1 182938-08-5, Rho-associated coiled-coil forming protein kinase II 182970-56-5, Matrix metalloproteinase 16 183257-54-7, Heparansulfate 3-sulfotransferase 184049-62-5, Dual-specificity protein phosphatase 6 185915-22-4, Fibroblast growth factor 13 186270-49-5, Angiopoietin 1 187042-29-1 188417-84-7, Vascular endothelial growth factor C 188596-65-8, Mitogen-activated protein kinase kinase kinase 14 189088-86-6, p21-Activated kinase 3 190606-22-5, Protein kinase 38 191359-14-5, MAP kinase-interacting kinase 2 191550-14-8, 8-Oxoguanine DNA glycosylase 192140-83-3, p21-Activated kinase 2 192230-91-4, Mitogen-activated protein kinase kinase 4 192588-76-4, CASP8 and FADD-like apoptosis regulator 192662-83-2, Vascular endothelial growth factor B 193830-48-7, Urocortin 197664-51-0, Protein kinase STK10 199877-11-7, Protein kinase PCTAIRE 2 202420-40-4, Gene STK11 protein kinase 203810-04-2, Protein kinase MRCK 206566-35-0, Molybdenum cofactor sulfurase 208778-50-1, Growth differentiation factor 9 214915-11-4, MASP-1 serine protease 219575-48-1, Ste20-like kinase 220064-77-7, p21-Activated protein kinase 4 223670-05-1, Apoptosis-associated tyrosine kinase 227018-38-4, Neuropilin 2 229977-92-8, PFTAIRE protein kinase 1 241824-56-6, Death-associated protein kinase 2 243859-94-1, Puromycin sensitive aminopeptidase 245524-76-9 252337-44-3 252852-50-9, SUMO-specific protease 252902-02-6, Homeodomain interacting protein kinase 2 271597-10-5, Growth differentiation factor 1 286383-12-8, Transmembrane serine protease 3 288307-53-9, Inositol 1,3,4-trisphosphate 5/6-kinase 289899-93-0, Mitogen-activatedproteinkinase9 291756-39-3, Mitogen-activatedproteinkinase10 293321-87-6, Metalloproteinase ADAM 23 296277-84-4, SNF1 related protein kinase 300570-67-6, Protein kinase H11 300857-98-1, Protein tyrosine phosphatase, receptor type, F 300858-62-2, Protein tyrosine phosphatase, receptor type, E 301166-54-1, Phosphataseandtensin homolog 306298-57-7, Dual-specificity protein phosphatase 9 321976-25-4, Sialyltransferase 322637-18-3, Fibroblast growth factor 18 329967-85-3 330197-29-0, Cyclin-dependentkinase 7 333425-95-9, Protein kinase D2 335605-46-4, Mitogen-activated protein kinase kinase 7 342900-44-1, Kallikrein 13

347894-56-8, p21-Activated protein kinase 6 353459-11-7, NIMA-related protein kinase 357400-48-7, Metalloproteinase ADAM 21 360565-62-4, Mitogen-activated protein kinase phosphatase x 361540-77-4 362479-32-1, Protein phosphatase 1, 362674-81-5, Protein phosphatase 2 366806-33-9, Casein kinase 2 367924-80-9, Integrin-linked kinase-associated serine/threonine phosphatase 2C 367950-11-6, MAP/microtubule affinity-regulating kinase3 386278-22-4, Death-associated protein kinase 3 388092-42-0, Prohormone convertase 2 400653-73-8, Dual specificity phosphatase 5 402736-19-0, Serum and glucocorticoid dependent kinase 2 402934-70-7, NIMA related kinase 7 403648-77-1, CDC28 protein kinase2 404344-49-6, Mitogen-activated protein kinase kinase kinase 3 405202-87-1, Mitogen-activated protein kinase kinase kinase 11 409105-92-6, Microtubule-associated testis-specific serine/threonine protein kinase 415715-09-2, BMP-2 inducible kinase 421595-36-0, Protein phosphatase 1D 424830-43-3, Prohormone convertase 5 428817-87-2, IRAK-4 kinase 436097-33-5, Vaccinia related kinase 2 440354-98-3, Cholesterol side-chain cleaving enzyme 443900-95-6, Glycogen synthase kinase 3β 443906-18-1, RPTP-κ 472998-88-2, Protein kinase Cι 475489-73-7, Calcium calmodulin-dependent protein kinase II 475678-93-4, WW domain containing oxidoreductase 644990-19-2, Peroxiredoxin 2 644990-62-5, Peroxiredoxin 3 644990-68-1, Peroxiredoxin 4 644991-16-2, Anti-oxidant protein 2 657407-83-5, Calpain 3
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gene for, expression of, in diagnosis of Alzheimer's disease; gene expression profiles in diagnosis and treatment of Alzheimer's disease)

L5 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2004:20538 CAPLUS Full-text
DN 140:89913
TI p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases
IN Doi, Hirofumi; Hosogi, Shinya; Wada, Naoya
PA Celestar Lexico-Sciences, Inc., Japan; Daiichi Pharmaceutical Co., Ltd.
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004002532	A1	20040108	WO 2003-JP8179	20030627
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003246091	A1	20040119	AU 2003-246091	20030627
	US 2006172360	A1	20060803	US 2005-519465	20050209
PRAI	JP 2002-190909	A	20020628		
	JP 2002-190910	A	20020628		
	WO 2003-JP8179	W	20030627		

AB This invention provides inhibitors of c-Jun phosphorylation by c-Jun NH2-terminal kinase 3 (JNK3) and a method of inhibiting binding of p21-activated kinase 4 (PAK4) to MKK7, and phosphorylation of MKK7 by PAK4, binding of JNK/SAPK-inhibitory kinase (JIK) to MKK7 and phosphorylation of MKK7 by JIK is inhibited; a preventive and/or a remedy for diseases mediated by c-Jun phosphorylation by JNK3. It also provide a method of identifying compound inhibiting binding of PAK4 to MKK7, phosphorylation of MKK7 by PAK4, binding of JIK to MKK7 and phosphorylation of MKK7 by JIK. It provide a medicinal composition containing the inhibitor compound identified. The authors predicted binding of MKK7 with PAK4 and JIK in silico and confirmed exptl. Activation of JNK3 signaling by phosphorylation of MKK7 by PAK4 and JIK was also found.

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases

AB This invention provides inhibitors of c-Jun phosphorylation by c-Jun NH2-terminal kinase 3 (JNK3) and a method of inhibiting binding of p21-activated kinase 4 (PAK4) to MKK7, and phosphorylation of MKK7 by PAK4, binding of JNK/SAPK-inhibitory kinase (JIK) to MKK7 and phosphorylation of MKK7 by JIK is inhibited; a preventive and/or a remedy for diseases mediated by c-Jun phosphorylation by JNK3. It also provide a method of identifying compound inhibiting binding of PAK4 to MKK7, phosphorylation of MKK7 by PAK4, binding of JIK to MKK7 and phosphorylation of MKK7 by JIK. It provide a medicinal composition containing the inhibitor compound identified. The authors predicted binding of MKK7 with PAK4 and JIK in silico and confirmed exptl. Activation of JNK3 signaling by phosphorylation of MKK7 by PAK4 and JIK was also found.

ST p21 kinase PAK4 phosphorylation MKK7 JNK3 signaling; JNK SAPK inhibitory kinase JIK phosphorylation MKK7 JNK3 signaling; neurodegenerative disease therapy PAK4 JIK MKK7 phosphorylation inhibitor

IT Brain, disease
Prion diseases
(Creutzfeldt-Jakob; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Brain, disease
Prion diseases
(Gerstmann-Straussler syndrome; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Nervous system, disease
(Huntington's chorea; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Mental and behavioral disorders
(Pick's disease; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Nervous system, disease
(amyotrophic lateral sclerosis, familial; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun

NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Muscle, disease
(atrophy; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Brain
(basal ganglia, cortex basal ganglion degenerative disease; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Transcription factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(c-jun, phosphorylation by JNK3, inhibition of; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Nervous system, disease
(degeneration; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Disease, animal
(degenerative, cortex basal ganglion; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Mental and behavioral disorders
(dementia, familial British; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Mental and behavioral disorders
(dementia, familial, associated with neuroserpin inclusion bodies; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Mental and behavioral disorders
(dementia, familial; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Mental and behavioral disorders
(diffuse Lewy body disease; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Brain, disease
Prion diseases
(mad cow; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Nervous system, disease
(multiple system atrophy; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and

phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Alzheimer's disease
Down's syndrome
Parkinson's disease
Signal transduction, biological
(p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Disease, animal
(polyglutamic; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Phosphorylation, biological
(protein; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Paralysis
(pseudobulbar; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Brain
(red nucleus, dentate nucleus red nucleus globous pallidus atrophy; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT Nervous system, disease
(spinocerebellar degeneration; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT 291756-39-3, JNK3
RL: BSU (Biological study, unclassified); BIOL (Biological study)
((c-Jun NH2-terminal kinase 3); p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT 220064-77-7, PAK4 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PAK4 (p21-activated kinase 4); p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT 253873-53-9, JNK/SAPK-inhibitory kinase 335605-46-4, MKK7 kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling: use in treatment of neurodegenerative diseases)

IT 642087-50-1 642087-55-6 642087-59-0 642087-64-7 642087-68-1
642087-71-6 642087-73-8 642087-77-2 642087-80-7 642087-82-9
642087-85-2 642087-88-5 642087-91-0 642087-94-3 642087-97-6
642088-00-4 642088-03-7 642088-06-0 642088-09-3 642088-12-8

642088-15-1 642088-18-4 642088-21-9 642088-23-1 642088-25-3
642088-27-5 642088-29-7 642088-31-1 642088-33-3 642088-35-5
642088-37-7 642088-39-9 642088-41-3 642088-43-5 642088-45-7
642088-46-8 642088-48-0 642088-50-4 642088-52-6 642088-54-8
642088-56-0 642088-58-2 642088-60-6 642104-96-9 642104-97-0
642104-98-1
RL: PRP (Properties)
(unclaimed sequence; p21-activated kinase 4 and JNK/SAPK-inhibitory kinase bind and phosphorylate MKK7 and activate c-Jun NH2-terminal kinase 3 signaling, use in treatment of neurodegenerative diseases)

L5 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 4
AN 2006:442464 BIOSIS Full-text
DN PREV200600443086
TI Taurine-responsive genes related to signal transduction as identified by cDNA microarray analyses of HepG2 cells.
AU Park, Sung-Hee; Lee, Haemi; Park, Kun Koo; Kim, Ha Won; Park, Taesun [Reprint Author]
CS Yonsei Univ, Dept Food and Nutr, Sudaemun Ku, 134 Shinchon Dong, Seoul 120749, South Korea
tspark@yonsei.ac.kr
SO Journal of Medicinal Food, (SPR 2006) Vol. 9, No. 1, pp. 33-41.
ISSN: 1096-620X.
DT Article
LA English
OS GenBank-AA909333; EMBL-AA909333; DDBJ-AA909333; GenBank-AA490664; EMBL-AA490664; DDBJ-AA490664; GenBank-U53174; EMBL-U53174; DDBJ-U53174; GenBank-AF029669; EMBL-AF029669; DDBJ-AF029669; GenBank-M81735; EMBL-M81735; DDBJ-M81735; GenBank-E00829; EMBL-E00829; DDBJ-E00829; GenBank-AAF039843; EMBL-AAF039843; DDBJ-AAF039843; GenBank-R23548; EMBL-R23548; DDBJ-R23548; GenBank-M35296; EMBL-M35296; DDBJ-M35296; GenBank-U03865; EMBL-U03865; DDBJ-U03865; GenBank-AA568151; EMBL-AA568151; DDBJ-AA568151; GenBank-AI500475; EMBL-AI500475; DDBJ-AI500475; GenBank-AI949483; EMBL-AI949483; DDBJ-AI949483; GenBank-Y11395; EMBL-Y11395; DDBJ-Y11395; GenBank-NM000735; EMBL-NM000735; DDBJ-NM000735; GenBank-U94905; EMBL-U94905; DDBJ-U94905; GenBank-X85106; EMBL-X85106; DDBJ-X85106
ED Entered STN: 6 Sep 2006
Last Updated on STN: 6 Sep 2006
AB Taurine-induced changes in the expression profiles of HepG2 cells were assessed using a cDNA microarray technology, and confirmed by real-time reverse transcription-polymerase chain reaction (RT-PCR) analyses. Of 8,298 human genes on the microarray, 128 genes (87 known genes) were up-regulated, and 349 (206 known genes) were down-regulated more than 2.0-fold by taurine. Among the 293 known genes regulated by taurine, a total of 44 genes were involved in signal transduction; 16 genes were up-regulated greater than 2.0-fold, and 28 genes were down-regulated more than 2.0-fold by taurine. The results of RT-PCR analyses for the five genes selected were consistent with our microarray data, although the fold changes in the expression level differed somewhat between the two analytical methods. Among signal transduction-related genes affected by taurine, four genes-mitogen-activated protein kinase (MAPK) kinase kinase 7, p21-activated kinase 4, sprouty homolog 2, and MAPK kinase I-are implicated in the MAPK signaling pathway. Taurine also regulated the expression of signal transducer and activator of transcription (STAT) 3 gene involved in the Janus kinase-STAT pathway, and diacylglycerol kinase, zeta 104 kDa, the downstream mediator of the protein kinase C transmembrane signaling pathway. In conclusion, gene expression profiling of HepG2 cells treated with taurine provided us with new insights

into the novel aspects of taurine as a possible regulator of MAPK signaling cascades and protein kinase C signaling pathways involved in cellular processes such as cell growth, differentiation, and apoptosis.

AB Taurine-induced changes in the expression profiles of HepG2 cells were assessed using a cDNA microarray technology, and confirmed by real-time reverse transcription-polymerase chain reaction (RT-PCR) analyses. Of 8,298 human genes on the microarray, 128 genes (87 known genes) were up-regulated, and 349 (206 known genes) were down-regulated more than 2.0-fold by taurine. Among the 293 known genes regulated by taurine, a total of 44 genes were involved in signal transduction; 16 genes were up-regulated greater than 2.0-fold, and 28 genes were down-regulated more than 2.0-fold by taurine. The results of RT-PCR analyses for the five genes selected were consistent with our microarray data, although the fold changes in the expression level differed somewhat between the two analytical methods. Among signal transduction-related genes affected by taurine, four genes-mitogen-activated protein kinase (MAPK) kinase kinase 7, p21-activated kinase 4, sprouty homolog 2, and MAPK kinase I-are implicated in the MAPK signaling pathway. Taurine also regulated the expression of signal transducer and activator of transcription (STAT) 3 gene involved in the Janus kinase-STAT pathway, and diacylglycerol kinase, zeta 104 kDa, the downstream mediator of the protein kinase C transmembrane signaling pathway. In conclusion, gene expression profiling of HepG2 cells treated with taurine provided us with new insights into the novel aspects of taurine as a possible regulator of MAPK signaling cascades and protein kinase C signaling pathways involved in cellular processes such as cell growth, differentiation, and apoptosis.

GEN human RAD9A gene (Hominidae): expression; human MAPK kinase 7 gene [human mitogen-activated protein kinase kinase 7 gene] (Hominidae): expression; human p21-activated kinase 4 gene (Hominidae): expression; human sprouty homolog 2 gene (Hominidae): expression; human MAPK kinase 1 gene [human mitogen-activated protein kinase kinase 1 gene] (Hominidae): expression; human STAT 3 gene [human signal transducer and activator of transcription 3 gene] (Hominidae): expression; human diacylglycerol kinase gene (Hominidae): expression; human zeta 104 kDa gene (Hominidae): expression; human XRCC4 gene (Hominidae): expression; human COPS2 gene (Hominidae): expression; human POLD1 gene (Hominidae): expression; human tachykinin receptor 1 gene (Hominidae); human adenosine A2a receptor gene (Hominidae); human G-protein coupled receptor 34 gene (Hominidae); human chimerin gene (Hominidae): expression

L5 ANSWER 8 OF 9 IFIPAT COPYRIGHT 2006 IFI on STN DUPLICATE 3
AN 11223312 IFIPAT;IFIUDB;IFICDB Full-text
TI MKK7 ACTIVATION INHIBITOR
INF Doi; Hirofumi, Chiba, JP
Hosogi; Shinya, Chiba, JP
Wada; Naoya, Tokyo, JP
IN Doi Hirofumi (JP); Hosogi Shinya (JP); Wada Naoya (JP)
PAF Unassigned
PA Unassigned Or Assigned To Individual (68000)
AG KILYK & BOWERSOX, P.L.L.C., 400 HOLIDAY COURT, SUITE 102, WARRENTON, VA, 20186, US
PI US 2006172360 A1 20060803
AI US 2003-519465 20030627
WO 2003-JP8179 20030627
20050209 PCT 371 date
20050209 PCT 102(e) date
PRAI JP 2002-190909 20020628
JP 2002-190910 20020628
FI US 2006172360 20060803

DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
CLMN 19
GI 8 Figure(s).

FIG. 1 illustrates the results of an in-silico prediction of the interaction of MKK7 with PAK4. Local alignment between MKK7 and PAK4 was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in MKK7 and those in PAK4, respectively.

FIG. 2 shows that MKK7 was phosphorylated in-vitro by PAK4. GSTMKK7 was phosphorylated in the presence of FLAG-PAK4 (lane 4), but not phosphorylated in the absence of FLAG-PAK4 (lane 3). On the other hand, GST was not phosphorylated either in the presence (lane 2) or the absence (lane 1) of FLAG-PAK4.-The values shown on the left-hand side of the figure represent molecular weights.

FIG. 3 shows that the binding of PAK4 to MKK7 was observed in a cell. The bottom panel of the figure shows the result of an immunoprecipitation test (IP), indicating that an immunoprecipitate containing HA-MKK7 and FLAG-PAK4 was detected in a cell lysate prepared from cells co-expressing HA-MKK7 and FLAG-PAK4 (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only HA-MKK7 (lane 1). The top and middle panels, respectively, show the results of verification of the expression of FLAG-PAK4 and HA-MKK7 in each cell lysate. Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 4 shows that the temporary expression of PAK4 increased cJun phosphorylation by JNK3 dependently on the amount of expressed PAK4. The bottom panel in the figure shows the result of a kinase assay, indicating that GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-PAK4 and FLAG-JNK3 (lanes 2-4), but not phosphorylated when using a cell lysate prepared from cells expressing only FLAG-JNK3 (lanes 1). Lanes 2, 3 and 4 show the results when transfecting with an HA-PAK4 expression vector (pcDNA-HA-PAK4) in amounts of 0.1 mu g, 0.5 mu g and 2.0 mu g, respectively. The top and middle panels show respectively the results of verification of the expression of FLAG-JNK3 and HAPAK4. The verification of expression was carried out by western blotting (WB).

FIG. 5 illustrates the results of an in-silico prediction of the interaction of MKK7 with JIK. Local alignment between MKK7 and JIK was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in MKK7 and those in JIK, respectively.

FIG. 6 shows that MKK7 was phosphorylated in-vitro by JIK. GSTMKK7 was phosphorylated in the presence of HA-JIK (lane 2), but not phosphorylated in the absence of HA-JIK (lane 1). On the other hand, GST was not phosphorylated in the presence (lane 3) of HA-JIK. The values shown on the left-hand side of the figure represent molecular weights.

FIG. 7 shows that the binding of JIK to MKK7 was observed in a cell. The bottom panel of the figure shows the result of immunoprecipitation test (IP), indicating that an immunoprecipitate containing FLAG-MKK7 and HA-JIK was detected in a cell lysate prepared from cells co-expressing FLAG-MKK7 and HA-JIK (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only FLAG-MKK7 (lane 1). The top and middle panels, respectively, show the results of the verification of expression of HA-JIK and FLAG-MKK7 in each cell lysate. Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 8 shows that the temporary expression of JIK increased cJun phosphorylation caused by JNK3. GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-JIK and FLAG-JNK3 (lanes 3), but not phosphorylated when using a cell lysate prepared from cells expressing neither HA-JIK nor FLAG-JNK3 (lane 1) or cells expressing only FLAG-JNK3 (lanes 2).

AB PAK4 and JIK, both of which bind to MKK7 and directly phosphorylate MKK7, were found in the present invention. The present invention provides an inhibitor of c-Jun phosphorylation caused by JNK3 and a method for inhibiting the same, and an agent for preventing and/or treating a disorder attributable to c-Jun phosphorylation caused by JNK3 and a method for preventing and/or treating the same, all of which comprise inhibiting one member selected from the following: the binding of PAK4 to MKK7, the phosphorylation of MKK7 by PAK4, the binding of JIK to MKK7, and the phosphorylation of MKK7 by JIK. Further, the present invention provides a method for identifying a compound that inhibits the binding of PAK4 to MKK7 , the phosphorylation of MKK7 caused by PAK4, the binding of JIK to MKK7, or the phosphorylation of MKK7 caused by JIK, as well as the compound obtained thereby. Furthermore, the present invention provides a pharmaceutical composition containing an effective amount of at least one member selected from the group consisting of the aforementioned compound and the aforementioned inhibitor.

CLMN 19 8 Figure(s).

FIG. 1 illustrates the results of an in-silico prediction of the interaction of MKK7 with PAK4. Local alignment between MKK7 and PAK4 was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in MKK7 and those in PAK4, respectively.

FIG. 2 shows that MKK7 was phosphorylated in-vitro by PAK4. GSTMKK7 was phosphorylated in the presence of FLAG-PAK4 (lane 4), but not phosphorylated in the absence of FLAG-PAK4 (lane 3). On the other hand, GST was not phosphorylated either in the presence (lane 2) or the absence (lane 1) of FLAG-PAK4.-The values shown on the left-hand side of the figure represent molecular weights.

FIG. 3 shows that the binding of PAK4 to MKK7 was observed in a cell. The bottom panel of the figure shows the result of an immunoprecipitation test (IP), indicating that an immunoprecipitate containing HA-MKK7 and FLAG-PAK4 was detected in a cell lysate prepared from cells co-expressing HA-MKK7 and FLAG-PAK4 (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only HA-MKK7 (lane 1). The top and middle panels, respectively, show the results of verification of the expression of FLAG-PAK4 and HA-MKK7 in each cell lysate. Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 4 shows that the temporary expression of PAK4 increased cJun phosphorylation by JNK3 dependently on the amount of expressed PAK4. The bottom panel in the figure shows the result of a kinase assay, indicating that GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-PAK4 and FLAG-JNK3 (lanes 2-4), but not phosphorylated when using a cell lysate prepared from cells expressing only FLAG-JNK3 (lanes 1). Lanes 2, 3 and 4 show the results when transfecting with an HA-PAK4 expression vector (pcDNA-HA-PAK4) in amounts of 0.1 mu g, 0.5 mu g and 2.0 mu g, respectively. The top and middle panels show respectively the results of verification of the expression of FLAG-JNK3 and HAPAK4. The verification of expression was carried out by western blotting (WB).

FIG. 5 illustrates the results of an in-silico prediction of the interaction of MKK7 with JIK. Local alignment between MKK7 and JIK was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in

MKK7 and those in JIK, respectively.

FIG. 6 shows that MKK7 was phosphorylated in-vitro by JIK. GSTMKK7 was phosphorylated in the presence of HA-JIK (lane 2), but not phosphorylated in the absence of HA-JIK (lane 1). On the other hand, GST was not phosphorylated in the presence (lane 3) of HA-JIK. The values shown on the left-hand side of the figure represent molecular weights.

FIG. 7 shows that the binding of JIK to MKK7 was observed in a cell. The bottom panel of the figure shows the result of immunoprecipitation test (IP), indicating that an immunoprecipitate containing FLAG-MKK7 and HA-JIK was detected in a cell lysate prepared from cells co-expressing FLAG-MKK7 and HA-JIK (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only FLAG-MKK7 (lane 1). The top and middle panels, respectively, show the results of the verification of expression of HA-JIK and FLAG-MKK7 in each cell lysate. Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 8 shows that the temporary expression of JIK increased cJun phosphorylation caused by JNK3. GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-JIK and FLAG-JNK3 (lanes 3), but not phosphorylated when using a cell lysate prepared from cells expressing neither HA-JIK nor FLAG-JNK3 (lane 1) or cells expressing only FLAG-JNK3 (lanes 2).

TI MKK7 ACTIVATION INHIBITOR

AB PAK4 and JIK, both of which bind to MKK7 and directly phosphorylate MKK7, were found in the present invention. The present invention provides an inhibitor of c-Jun phosphorylation caused by JNK3 and a method for inhibiting the same, and an agent for preventing and/or treating a disorder attributable to c-Jun phosphorylation caused by JNK3 and a method for preventing and/or treating the same, all of which comprise inhibiting one member selected from the following: the binding of PAK4 to MKK7, the phosphorylation of MKK7 by PAK4, the binding of JIK to MKK7, and the phosphorylation of MKK7 by JIK. Further, the present invention provides a method for identifying a compound that inhibits the binding of PAK4 to MKK7 , the phosphorylation of MKK7 caused by PAK4, the binding of JIK to MKK7, or the phosphorylation of MKK7 caused by JIK, as well as the compound obtained thereby. Furthermore, the present invention provides a pharmaceutical composition containing an effective amount of at least one member selected from the group consisting of the aforementioned compound and the aforementioned inhibitor.

GI 8 Figure(s).

FIG. 1 illustrates the results of an in-silico prediction of the interaction of MKK7 with PAK4. Local alignment between MKK7 and PAK4 was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in MKK7 and those in PAK4, respectively.

FIG. 2 shows that MKK7 was phosphorylated in-vitro by PAK4. GSTMKK7 was phosphorylated in the presence of FLAG-PAK4 (lane 4), but not phosphorylated in the absence of FLAG-PAK4 (lane 3). On the other hand, GST was not phosphorylated either in the presence (lane 2) or the absence (lane 1) of FLAG-PAK4.-The values shown on the left-hand side of the figure represent molecular weights.

FIG. 3 shows that the binding of PAK4 to MKK7 was observed in a cell. The bottom panel of the figure shows the result of an immunoprecipitation test (IP), indicating that an immunoprecipitate containing HA-MKK7 and FLAG-PAK4 was detected in a cell lysate prepared from cells co-expressing HA-MKK7 and FLAG-PAK4 (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only HA-MKK7 (lane 1). The top and middle panels, respectively, show the results of verification of the expression of FLAG-PAK4 and HA-MKK7 in each cell lysate.

Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 4 shows that the temporary expression of PAK4 increased cJun phosphorylation by JNK3 dependently on the amount of expressed PAK4. The bottom panel in the figure shows the result of a kinase assay, indicating that GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-PAK4 and FLAG-JNK3 (lanes 2-4), but not phosphorylated when using a cell lysate prepared from cells expressing only FLAG-JNK3 (lanes 1). Lanes 2, 3 and 4 show the results when transfecting with an HA-PAK4 expression vector (pcDNA-HA-PAK4) in amounts of 0.1 mu g, 0.5 mu g and 2.0 mu g, respectively. The top and middle panels show respectively the results of verification of the expression of FLAG-JNK3 and HAPAK4. The verification of expression was carried out by western blotting (WB).

FIG. 5 illustrates the results of an in-silico prediction of the interaction of MKK7 with JIK. Local alignment between MKK7 and JIK was conducted, and regions with high scores are shown. The upper and lower rows indicate partial sequences present in MKK7 and those in JIK, respectively.

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FIG. 7 shows that the binding of JIK to MKK7 was observed in a cell. The bottom panel of the figure shows the result of immunoprecipitation test (IP), indicating that an immunoprecipitate containing FLAG-MKK7 and HA-JIK was detected in a cell lysate prepared from cells co-expressing FLAG-MKK7 and HA-JIK (lane 2), though such an immunoprecipitate was not detected in a cell lysate prepared from cells expressing only FLAG-MKK7 (lane 1). The top and middle panels, respectively, show the results of the verification of expression of HA-JIK and FLAG-MKK7 in each cell lysate. Detection of the immunoprecipitate and verification of the expression were both carried out by western blotting (WB).

FIG. 8 shows that the temporary expression of JIK increased cJun phosphorylation caused by JNK3. GST-c-Jun(1-79) was phosphorylated when using a cell lysate prepared from cells coexpressing HA-JIK and FLAG-JNK3 (lanes 3), but not phosphorylated when using a cell lysate prepared from cells expressing neither HA-JIK nor FLAG-JNK3 (lane 1) or cells expressing only FLAG-JNK3 (lanes 2).

ECLM .D R A W I N G

1. An inhibitor of c-Jun phosphorylation caused by c-Jun Nterminal kinase 3, having at least one function selected from the group consisting of the following functions: i) inhibiting the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7); ii) inhibiting the phosphorylation of MKK7 caused by PAK4; iii) inhibiting the binding of JNK/SAPK-inhibitory kinase (JIK) to MAP kinase kinase 7 (MKK7); and iv) inhibiting the phosphorylation of MKK7 caused by JIK.

ACLM 2. A method for inhibiting c-Jun phosphorylation caused by c-Jun N-terminal kinase 3, comprising at least one step selected from the group consisting of the following steps: i) inhibiting the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7); ii) inhibiting the phosphorylation of MKK7 caused by PAK4; iii) inhibiting the binding of JNK/SAPK-inhibitory kinase (JIK) to MAP kinase kinase 7 (MKK7

); and iv) inhibiting the phosphorylation of MKK7 caused by JIK.

3. An agent for preventing and/or treating a disorder attributable to c-Jun phosphorylation caused by c-Jun N-terminal kinase 3, having at least one function selected from the group consisting of the following functions i) inhibiting the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7); ii) inhibiting the phosphorylation of MKK7 caused by PAK4; iii) inhibiting the binding of JNK/SAPK-inhibitory kinase (JIK) to MAP kinase kinase 7 (MKK7); and iv) inhibiting the phosphorylation of MKK7 caused by JIK.

5. A method for preventing and/or treating a disorder attributable to c-Jun phosphorylation caused by c-Jun N-terminal kinase 3, comprising at least one step selected from the group consisting of the following steps: i) inhibiting the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7); ii) inhibiting the phosphorylation of MKK7 caused by PAK4; iii) inhibiting the binding of JNK/SAPK-inhibitory kinase (JIK) to MAP kinase kinase 7 (MKK7); and iv) inhibiting the phosphorylation of MKK7 caused by JIK.

7. A method for identifying a compound that inhibits the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7), comprising contacting PAK4 and/or MKK7 with a test compound under conditions that allow the binding of PAK4 to MKK7; and determining whether the test compound inhibits the binding of PAK4 to MKK7, by detecting the presence, absence or change of a signal generated by the binding of PAK4 to MKK7.

8. A method for identifying a compound that inhibits the binding of JNK/SAPK-inhibitory kinase (JIK) to MAP kinase kinase 7 (MKK7), comprising contacting JIK and/or MKK7 with a test compound under conditions that allow the binding of JIK to MKK7; and determining whether the test compound inhibits the binding of JIK to MKK7, by detecting the presence, absence or change of a signal generated by the binding of JIK to MKK7.

9. A method for identifying a compound that inhibits the phosphorylation of MAP kinase kinase 7 (MKK7) caused by p21-activated kinase 4 (PAK4), comprising contacting PAK4 and/or MKK7 with a test compound; and determining whether the test compound inhibits the phosphorylation of MKK7 caused by PAK4, by introducing a system using a signal and/or a marker capable of detecting the phosphorylation of MKK7 and detecting the presence, absence or change of the signal and/or the marker.

10. A method for identifying a compound that inhibits the phosphorylation of MAP kinase kinase 7 (MKK7) caused by JNK/ SAPK-inhibitory kinase (JIK), comprising contacting JIK and/or MKK7 with a test compound; and determining whether the test compound inhibits the phosphorylation of MKK7 caused by JIK, by introducing a system using a signal and/or a marker capable of detecting the phosphorylation of MKK7 and detecting the presence, absence or change of the signal and/or the marker. 11-19. (canceled)

20. A pharmaceutical composition containing an effective amount of at least one member selected from the group consisting of the following compounds and the inhibitors: i) a compound that inhibits the binding of p21-activated kinase 4 (PAK4) to

MAP kinase kinase 7 (MKK7
), ii) a compound that inhibits the binding of JNK/SAPK-inhibitory kinase (JIK) to MKK7, iii) a compound that inhibits the phosphorylation of MKK7 caused by PAK4, iv) a compound that inhibits the phosphorylation of MKK7 caused by JIK, v) an inhibitor of the binding of PAK4 to MKK7, vi) an inhibitor of the binding of JIK to MKK7, vii) an inhibitor of the phosphorylation of MKK7 caused by PAK4; and viii) an inhibitor of the phosphorylation of MKK7 caused by JIK.

21. The agent for preventing and/or treating a disorder according to claim 3, wherein the agent contains an effective amount of at least one member selected from the group consisting of the following compounds and the inhibitors: i) a compound that inhibits the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7), ii) a compound that inhibits the binding of JNK/SAPK-inhibitory kinase (JIK) to MKK7, iii) a compound that inhibits the phosphorylation of MKK7 caused by PAK4, iv) a compound that inhibits the phosphorylation of MKK7 caused by JIK, v) an inhibitor of the binding of PAK4 to MKK7, vi) an inhibitor of the binding of JIK to MKK7 vii) an inhibitor of the phosphorylation of MKK7 caused by PAK4; and viii) an inhibitor of the phosphorylation of MKK7 caused by JIK.

24. The method for preventing and/or treating a disorder according to claim 5, comprising using at least one member selected from the group consisting of the following compounds and the inhibitors: i) a compound that inhibits the binding of p21-activated kinase 4 (PAK4) to MAP kinase kinase 7 (MKK7), ii) a compound that inhibits the binding of JNK/SAPK-inhibitory kinase (JIK) to MKK7, iii) a compound that inhibits the phosphorylation of MKK7 caused by PAK4, iv) a compound that inhibits the phosphorylation of MKK7 caused by JIK v) an inhibitor of the binding of PAK4 to MKK7, vi) an inhibitor of the binding of JIK to MKK7 vii) an inhibitor of the phosphorylation of MKK7 caused by PAK4; and viii) an inhibitor of the phosphorylation of MKK7 caused by JIK.

27. A reagent kit containing at least one member selected from the group consisting of p21-activated kinase 4 (PAK4), JNK/SAPK-inhibitory kinase (JIK), a polynucleotide encoding PAK4, a polynucleotide encoding JIK, a vector containing a polynucleotide encoding PAK4 and a vector containing a polynucleotide encoding JIK; and at least one member selected from the group consisting of MAP kinase kinase 7 (
MKK7), a polynucleotide encoding MKK7 and a vector containing a polynucleotide encoding MKK7.

L5 ANSWER 9 OF 9 USPATFULL on STN
AN 2004:44501 USPATFULL Full-text
TI Proteins and nucleic acids encoding same
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PI US 2004033493 A1 20040219
AI US 2002-72012 A1 20020131 (10)
PRAI US 2001-267459P 20010208 (60)
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US 2001-330245P 20011018 (60)
US 2001-332701P 20011114 (60)
US 2001-271664P 20010226 (60)
DT Utility
FS APPLICATION
LREP Ivor R. Elrifi, Ph.D., Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 59681
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed herein are nucleic acid sequences that encode novel polypeptides. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivatives, variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	318.31	352.08

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.00	-3.00

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